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Case report

Chronic mould exposure as a risk factor for severe community acquired pneumonia in a patient requiring extra corporeal membrane oxygenation

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A B S T R A C T

A previously fit and well man developed acute respiratory failure due to environmental mould exposure from living in damp rental accommodation. Despite aggressive intensive care management he rapidly deteriorated and required respiratory and cardiac Extracorporeal Membrane Oxygenation. We hypothesize that poor domiciliary conditions may make an underestimated contribution to community respiratory disease. These conditions may present as acute and severe illness with non-typical pathogens identified.

Introduction

Extracorporeal Membrane Oxygenation (ECMO) is a technique for providing respiratory and cardiac support to patients with severe acute pulmonary or cardiac failure that is potentially reversible but unresponsive to conventional management. ECMO provides oxygenation and rests the lungs, thereby decreasing the insult caused by mechanical ventilation [1]. The subject of this case developed severe respiratory failure due to chronic mould exposure from living in damp dwellings. Despite mechanical ventilation he deteriorated and required ECMO support. To our knowledge such severe community acquired pneumonia (CAP) secondary to chronic mould exposure has not previously been reported.

Case

A previously fit and well 31-year old warehouse worker presented with a 48-h history of shortness of breath, fever, chills and haemoptysis. On admission (day 0), he was diagnosed with severe community acquired pneumonia and was transferred to Intensive Care for mechanical ventilation. He rapidly deteriorated both clinically and radiologically and by day +2 he required immediate transfer to our adult Severe Respiratory Failure Unit (SRFU) for respiratory (veno-venous) Extracorporeal Membrane Oxygenation (ECMO) (see Fig. 1). He remained cardiovascularly unstable requiring increasing amounts of inotropes and was diagnosed with acute cardiomyopathy thought secondary to sepsis. An intra aortic balloon pump was inserted. Despite this intervention, continuing cardiac instability meant that on day +3 he was converted to cardiac (veno-artrial) ECMO.

A full septic screen was carried out on admission which included a *Legionella* and *Streptococcus pneumoniae* urinary antigen test, an atypical serology test (including mycoplasma and chlamydophila), a viral PCR screen for respiratory viruses (Influenza A&B, Parainfluenza 1,2&3, RSV, Metapnuemovirus, Adenovirus and Rhinovirus) and an HIV test. All admission tests were negative. However, on day +1 communication from the referring hospital reported that blood cultures, a tracheal aspirate and pleural fluid, collected at the referring hospital (day 2), had grown *Acinetobacter baumannii*. A detailed social history taken from the patient's wife reported that for the past 4 years the family has lived in rented accommodation which was noticeably damp and mouldy. Over that period of time,

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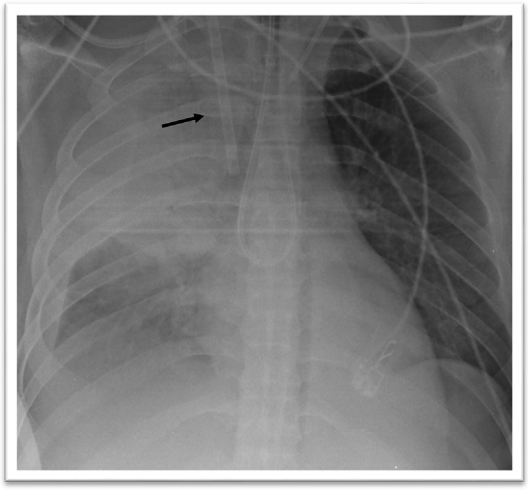

Fig. 1. Chest X ray on admission to the SRFU for ECMO. Black arrow indicates the position of the respiratory (VV) ECMO cannula.

her husband often complained of sleep disturbances due to an asthma-like-illness and two of their five children had recently been hospitalized secondary to a viral illness.

In view of this information, the family home was referred to the Mould Surveillance Service (MSS) run by the Mycology Reference Centre based at the University Hospital of South Manchester. The air (one cubic meter per room) in the patient's bedroom, an adjoining bathroom and children's bedrooms, and the lounge area were sampled using a SAS Duo 360 High Volume Microbial Air Sampler (Cherwell Laboratories, Bicester, UK) following a protocol specified in ISO 16000-18: 2011. Fungal propagules were impacted onto malt extract agar. The culture plates were incubated at 30 °C for 96 h and then the colonies were enumerated and identified by microscopy. The home survey carried out found an extremely high level of mixed moulds, predominantly *Penicillium glabrum* species, in the air of the ground floor accommodation (see Figs. 2 and 3). In addition, all locations in the patient's house far exceeded the UK/European/WHO recommended threshold of 200–500 colony forming units per cubic meter of indoor air for any one mould species [2].

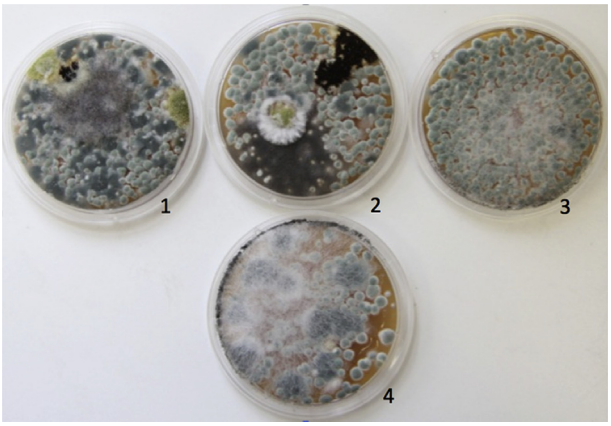

Fig. 2. Locations and sample type for mould surveillance cultures carried out at the patent's home. 1. Master bedroom (patient's bedroom), air sample: >1300 colony forming units (CFU) per cubic meter of air *Penicillium glabrum*, lower levels of *Aspergillus fumigatus*, *Aspergillus niger*, *Aspergillus flavus* and *Rhizopus oryzae*. 2. Adjacent bathroom (had undergone renovation): >1300 CFU mixed moulds. 3. Twins bedroom (adjacent to parent's bedroom and bathroom): >1300 CFU mixed moulds. 4. Lounge (where patient would go and sleep when feeling unwell): >1300 CFU mixed moulds.

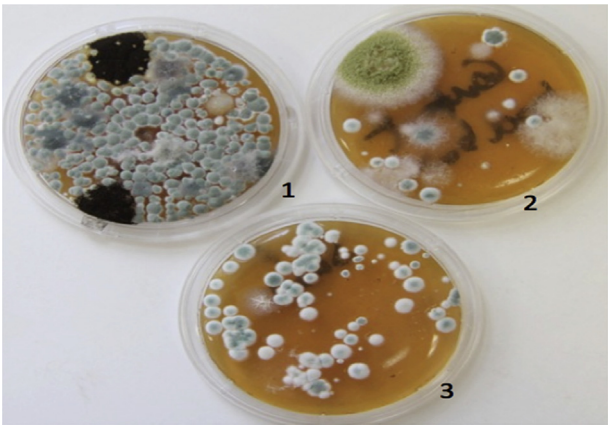

Fig. 3. Further sampling locations with sample type. 1. Inside patient's bed (a double storage type bed box): >1300 CFU mixed moulds. 2. Fabric base of box storage under bed, contact plate: light growth mixed moulds: *Penicillium glabrum*, *Aspergillus terreus*, *Aspergillus fumigatus* and *Geotrichum candidum*. 3. Mouldy shoe stored in bed box (previously stored in the wardrobe in the patient's bedroom), contact plate: predominantly *Penicillium glabrum*.

The patient was treated with meropenem, ciprofloxacin, high dose micafungin and prophylactic acyclovir (as per unit policy). He remained on ECMO for 10 days and in our unit for a further 8 days before improving enough for repatriation to his referring hospital at day+20, where he underwent Intensive Care rehabilitation and follow up by the cardiac and respiratory teams. In view of the fact that the patient had been exposed to this level of mould allergens for a period of four years, he has been referred to an outpatient respiratory infection clinic based in the National Aspergillosis Centre, where he will undergo a series of testing for inflammatory and allergic biomarkers.

Discussion

A review of studies in several European countries, Canada and the United States in 2004 indicated that at least 20% of buildings had one or more signs of dampness [2]. Dampness was defined on the basis of indicators such as water leakage or damage, bubbles or discolouration of floor coverings and visible mould growth indoors on walls, floors or ceilings. It is known that dampness can lead to growth of mould, fungi and bacteria, which emit spores, cells, fragments and volatile organic compounds into indoor air [3]. Dampness has therefore been suggested to be a strong, consistent indicator of risk of asthma and respiratory symptoms (e.g. cough and wheeze) and has been implicated in the causation of hypersensitivity pneumonitis (extrinsic allergic alveolitis) [3].

We report a previously fit and well young man who presented with such severe acute community acquired pneumonia that he required respiratory (and cardiac) ECMO. ECMO is used to provide respiratory and cardiac support oxygen to patients with severe pulmonary or cardiac failure that is refractory to mechanical ventilation. In our hospital the service was set up to manage patients with severe Influenza A infection as part of the Influenza Pandemic Plan. As technology and experience has advanced, indications for ECMO have broadened and ECMO is now available as a rescue therapy for many severe acute respiratory illnesses.

The patient in this case worked in a warehouse and undertook heavy lifting on a regular basis and so, despite being a heavy

smoker, was unlikely to have been previously diagnosed with a cardiac or respiratory condition. We hypothesize that chronic mixed mould exposure over a 4 year period, as a result of his domiciliary condition, had induced undiagnosed lung damage. This hypothesis is supported by environmental samples reporting high levels of mould contamination far exceeding international standards. In addition the primary pathogen (*Acinetobacter baumannii*), is an uncommon cause of community acquired pneumonia in someone with no underlying lung disease and suggestive of a more chronic (undiagnosed) condition. A damp indoor environment has also been associated with the survival of viruses so that the occupants are at greater risk of respiratory infection [4]. This may explain the recent viral exacerbations in the children.

Environmental moulds are known to pose a respiratory health risk in susceptible populations and chronic exposure to *Penicillium* species is known to increase exacerbations of asthma symptoms [3]. The home survey carried out by the MMS reported visible mould in the bedroom accommodation and under his mattress (which according to his wife had been recently turned). Mattresses constitute an important reservoir for mould, with measured concentrations of 10^3–10^7 spores/g of dust [5]. The recent physical disturbance may have led to aerosolization and subsequent inhalation of large quantities of fungal matter and this may have been the mechanism to trigger an acute respiratory deterioration.

Conclusion

We suggest that domiciliary conditions may make an underestimated contribution to community respiratory disease. These conditions may present as acute and severe illness (in this case requiring ECMO support), with atypical pathogens identified. The potential for chronic mould exposure as a risk factor for severe community acquired pneumonia must be recognized. Prevention of the conditions that lead to the adverse exposure in this case should be given priority to avoid additional contributions to poor health in the general population.

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